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Case Report

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Causation or coincidence? A case of methanol intoxication presenting with acute myocardial infarction and visual impairment

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Abstract

Methanol is a toxic alcohol component found in various household and industrial products. Methanol poisoning is a clinical disease with high morbidity and mortality rates. Patients often present to the emergency department with complaints of depression of the central nervous system, visual impairment, stomach symptoms, vomiting, and nausea, but cardiovascular complications occur less frequently. Here, we describe a case of methanol poisoning in which an acute myocardial infarction and visual impairment were combined.

Keywords: methanol intoxication, myocardial infarction, blindness, complication

1. Introduction

Methanol is a highly toxic, clear, and colorless alcohol similar to ethanol, which can cause poisoning by contact with the skin, ingestion, or even inhalation. Methanol is not cytotoxic, but its metabolic products are responsible for its toxicity. Methanol is metabolized to formaldehyde and then to formic acid. These metabolites alter oxygen utilization, inhibiting the mitochondrial cytochrome oxidase system, leading to "histotoxic hypoxia", resulting in metabolic acidosis, blindness, cardiovascular instability, and mortality (1).

The finding of methanol poisoning often occurs 12 to 24 hours after oral ingestion. This delay is related to the slow metabolism of methanol to its toxic metabolites. The patient may have visual problems, abdominal pain, vertigo, nausea, vomiting, and headache in the early period. If not treated in the late period, coma, blindness, gastrointestinal bleeding, putaminal bleeding, and death can develop (1, 2). Although neurological, ocular, metabolic, and gastrointestinal findings of methanol poisoning are well known, cases related to its cardiovascular effects are limited. A few cases of methanol poisoning were reported with acute coronary syndrome (3). Here, we share the clinical course and treatment of an intoxication case with myocardial infarction and visual loss.

2. Case Presentation

A 48-year-old male patient applied to the emergency service with the complaint of epigastric pain and blurred vision. The orientation and cooperation of the patient were weak. His blood pressure (90/50 mmHg) and oxygen saturation (%89) were low. On physical examination, both of his pupillary were dilated, and his reaction to light was markedly decreased. His visual acuity could not be evaluated due to agitation and non-cooperation. The electrocardiography (ECG) demonstrated elevation of ST in leads D2, D3, and aVF, consistent with acute inferior myocardial infarction (Fig. 1a). The blood gas was compatible with metabolic acidosis (pH:7.09, pCO_2:21 mmHg, pO_2: 97 mmHg, HCO_3: 5.9 mEq/dL). On biochemical examination, blood urea nitrogen (BUN) and creatinine were 50 mg/dl and 1.5 mg/dl, respectively. Cardiac troponin I (11,00 ng/ml) and Creatine Kinase-MB (CK-MB) (98,0 ng/dl) levels were elevated. The anamnesis taken from his relatives showed that he did not have any chronic diseases. He was informally producing alcohol and consumed illicit alcohol two days ago. As he had a respiratory arrest in the emergency service, he was intubated and initiated invasive mechanical ventilation. Metabolic acidosis with a wide anion gap and visual loss due to illicit alcohol consumption suggested methanol poisoning. The blood methanol level could not be checked because it was not examined in our hospital. The patient was administered parenteral intravenous saline and bicarbonate infusion with 10 mEq/h and taken to the angiography laboratory. In coronary angiography, there were non-critical atherosclerotic plaques in the left anterior descending artery (LAD) and the circumflex artery (Cx), while 100% thrombosis in the proximal part of the

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right coronary artery (RCA) (Fig. 1b). He received a 600 mg loading dose of clopidogrel and 300 mg of acetylsalicylic acid from the nasogastric catheter and 7500 units of heparin by intravenous injection (IV). A stent was successfully implanted into his proximal RCA (Fig. 1c). Fomepizole, an antidote used to treat poisoning with methanol or ethylene glycol, could not be administered during the patient's stay in the intensive care unit. 10% ethanol was administered with an IV loading dose of 10ml/kg, followed by a maintenance dose of 1 ml/kg/hour. The goal of therapy was a blood ethanol concentration between 120 and 140 mg/dl. During follow-up, the patient underwent two sessions of hemodialysis. The ethyl alcohol treatment was stopped when there was no acidosis in the control blood gas, and the anion gap was within the normal limits.

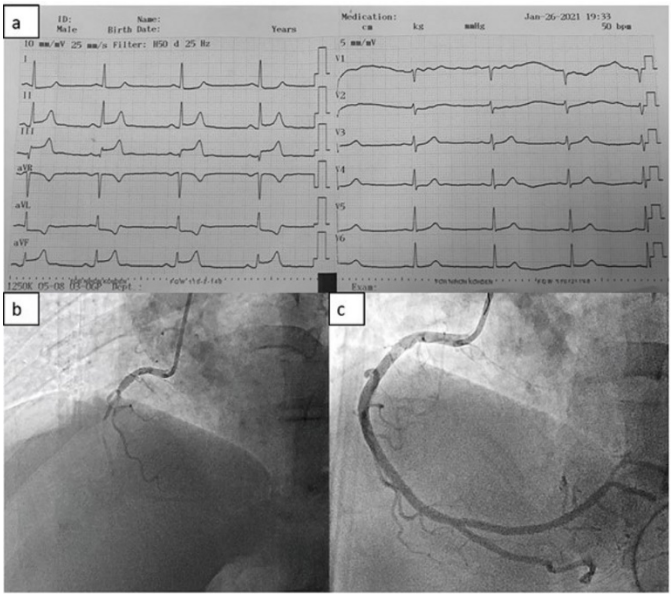

Fig. 1. (a): Electrocardiogram showed ST-segment elevation in D2-D3-aVF derivations and reciprocal ST depression in D1-aVL derivations. **(b):** First angiographic view of the occluded Right coronary artery (RCA). **(c):** Angiographic view of the RCA after percutaneous coronary intervention with stent

Neurological imaging was performed in the first 24 hours after the stabilization of the patient. Brain computed tomography (CT) showed hypodense areas in the bilateral putamen (Fig. 2a). Brain magnetic resonance imaging (MRI) showed hypointense areas in the T1-weighted axial sequence in the bilateral putamen and hyperintense areas in the T2-weighted axial sequence (Fig. 2b-c). The neurological analysis supported the diagnosis of methanol intoxication. Due to the complaint of blurred vision, intravenous methylprednisolone, vitamin B, and 25 mg of folic acid were started after an eye consultation. The patient was extubated on the third day of hospitalization. On ophthalmic examination performed after clinical stability, visual acuity was light sensation in the left eye and no light sensation in the right eye. On fundoscopic examination, both optic discs were pale in appearance (Fig. 3). There was no significant improvement in patient vision loss, while stable in terms of cardiovascular disease, and he was discharged 12 days after admission. Information about the subsequent clinical status of the patient could not be obtained because he did not reapply to our hospital after discharge. Written informed consent was obtained from the patient to publish this case report.

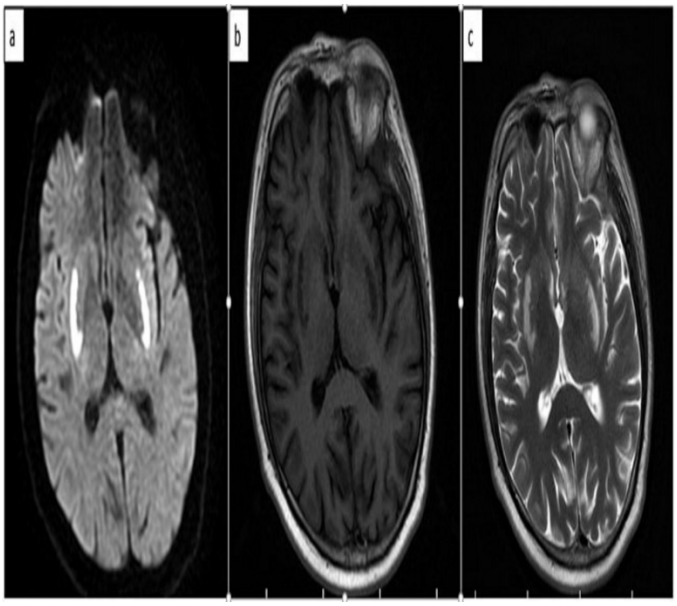

Fig. 2. (a): Brain MR image without contrast showing bilateral symmetrical changes in the putamen. Hypo intensity in T1W axial image. **(b):** Hyperintensity signal changes in T2W axial image. **(c):** Diffusion restriction in DW axial image

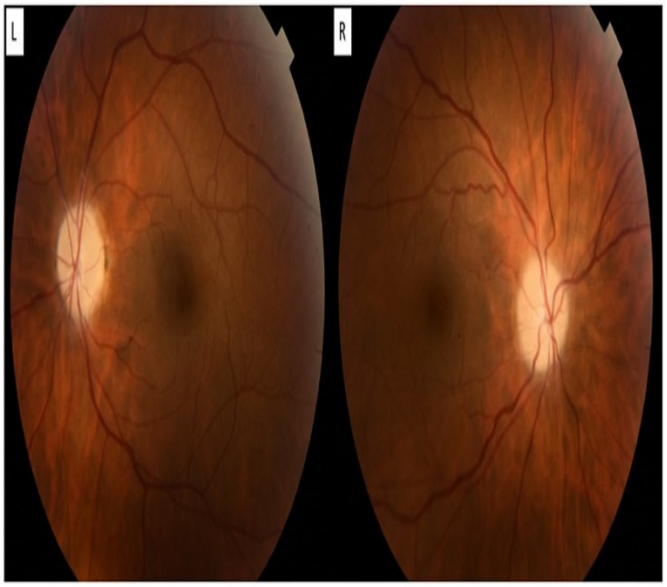

Fig. 3. Fundus photography showing optic disc pallor in the right and left eye

3. Discussion

Methanol intoxication is diagnosed by the history and characteristic neuroophthalmological findings and metabolic acidosis with a wide anion gap. The confirmation of the diagnosis is based on the demonstration of methanol in the blood; however, blood methanol analysis is not available in all centers. In our patient, the methanol level could not be checked. Due to his illicit alcohol consumption, characteristic clinical features, and high anion gap metabolic acidosis, he was

diagnosed with methanol poisoning. The first symptoms are usually nonspecific, and clinical manifestations become apparent after a latent period. As metabolic acidosis deepens, severe visual impairments, neurological symptoms, respiratory failure, and coma can develop and may even lead to death (1). Retinal ganglion cells and optic nerve axons are the primary targets of the neurotoxic results of formic acid (4). The clinical symptoms of methanol-induced optic neuropathy range from blurred or blacked-out vision, glare, photophobia, visual field defects, and total blindness. The patient expresses visual defects such as seeing a blizzard or total loss of light perception. The funduscopic examination may be normal in the early period or show optic disc hyperemia or pallor, venous enlargement, peripapillary edema, and retinal or optic disc edema. The pallor of the optical disc is an indicator of atrophy and is seen in the late period (5). Central nervous system symptoms can range from headache, dizziness, lethargy, confusion, and epileptic seizures to coma. In some cases, Parkinson's-like extrapyramidal syndrome may develop, such as rigidity, bradykinesia, mild tremor, and a masked face (1, 6). The most typical radiological findings in the toxicity of methanol are bilateral putaminal necroses that can have various degrees of bleeding. Putaminal necrosis and bleeding are likely due to the direct toxic influences of methanol metabolites and metabolic acidosis in the basal ganglia (7).

Methanol poisoning can cause cardiac dysfunction with various electrocardiographic abnormalities, but data on cardiovascular effects are limited. Although the direct cardiovascular effects of methanol or its metabolites are unknown, metabolic acidosis is generally thought to be responsible. Recently, in a study in which 356 methanol intoxications were examined in Iran, the most common ECG findings were elevation of the J point, U wave, early repolarization, QTc prolongation, and fragmented QRS. Furthermore, this study found myocardial infarction in 5.3% of the cases (8). In the literature, there are a limited number of cases of myocardial infarction accompanying methanol poisoning (3). Metabolic acidosis is known to reduce cardiac contractility and output, leading to hypotension, and has been shown to have arrhythmogenic effects in animal experiments (9). Methanol has also been shown to cause cardiac dilatation and myocyte degeneration in animal models. The pathophysiology of myocardial infarction is not exactly known. The possible mechanism may be endothelial dysfunction due to acidosis. The effects of acidosis on endothelial function are known. The impairment of endothelial function and altered inflammatory processes can increase thrombotic activity. Additionally, hypotension that develops with the vasodilator effect of acidosis and hemodynamic instability caused by the increased sympathetic activity to compensate may cause rupture of atherosclerotic plaques. Such hypotheses require further investigation and research.

Therapy choices for methanol poisoning cover supportive care, fomepizole, ethanol, dialysis, and, theoretically, folate. Fomepizole is more easily dosed, does not cause drunkenness, and inhibits alcohol dehydrogenase, but it is so expensive. Ethanol is cheaper but more difficult to dose accurately, requires close monitoring of serum ethanol concentration, and causes inebriation. Ethanol can also be used therapeutically to inhibit alcohol dehydrogenase when fomepizole is not available. Dialysis indications include serious metabolic acidosis, acute kidney failure, vision defects, electrolyte disorders, negative response to treatment, and central nervous system involvement (10).

Physicians should be aware of the possibility of methanol poisoning in patients with visual defects, increased metabolic acidosis of the anion gap, and change of consciousness. Although the cardiac presentation is not as common as ocular, gastrointestinal, and neurologic manifestations, it should be kept in mind when methanol poisoning is suspected. In methanol poisoning, patients may be unable to identify chest pain due to impaired consciousness. For this reason, routine cardiac evaluation is important in suspected patients.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report.

Conflict of interest

The authors declare no conflict of interest.

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Authors' contributions

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